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Electrocardiographic Changes Following Percutaneuos Alcohol Septal Ablation for Hypertrophic Cardiomyopathy

Garbor F. Matos, Michael Tempelhof, Krystyna Malinowska, Ferdinand S. Leya, Daniel J. Newton, John F. Moran and Leslie Cho Department of Medicine/Division of Cardiology, 2160 South First Avenue, Loyola University Health System, Maywood, Illinois 60153

Abstract: Percutaneous Transluminal Alcohol Septal Ablation (PTSA) of the hypertrophied septum for patients with Hypertrophic Obstructive Cardiomyopathy (HOCM) is an effective and safe endovascular non-surgical therapeutic intervention. We report a detailed analysis of Electrocardiographic (ECG) changes associated with PTSA immediately and a mean of 164 days after the procedure in 25 patients. A subgroup of 9 patients had long term follow up with mean of 360 days (108-783days). We correlated the ECG changes with hemodynamic and structural measurements on serial echocardiograms. We retrospectively reviewed the records of 25 patients who underwent PTSA for symptomatic HOCM in our tertiary center between 3/2001 and 5/2003. All patients had ECGs and echocardiograms pre, immediately post-PTSA and at follow-up evaluations. All patients had improvement in left ventricular outflow tract gradient, interventricular septal thickness and left atrial diameter. Baseline NYHA functional class of 2.8±0.7 improved post-PTSA to 1.1±1.0 ($p<0.001$). None of the patients experienced subsequent angina. One patient developed progressive heart failure and died 1-month post-PTSA of unclear etiology. Four (16%) patients required permanent dual chamber pacemaker implantation subsequent to the development of pacemaker dependent third degree heart block. Twelve (48%) patients developed right bundle branch block, 6 (24%) patients left anterior fascicular hemiblock, 1 (4%) patients left posterior fascicular hemiblock and no patients had new left bundle branch block. QRS duration of 113.4±32.1 msec increased 23% to 140.3±27.9 msec ($p<0.0001$) immediate post-PTSA. QTc interval prolonged from the baseline of 453.9±37.3 msec to 491.3±52.6 msec ($p<0.016$) immediately post-PTSA. Nine patients had over 3-month follow-up with QTc intervals of 460.7±44.9 msec pre- and 552.6±47.8 msec ($p<0.005$) immediately post-PTSA. At last follow-up QTc interval decreased to 487.7±41.4 msec, which was not significantly different from the baseline. JTc interval showed no significant change from baseline immediately post PTSA or at long term follow up. New, at least 1 additional mm ST segment elevation developed in any of the anterior chest leads in four patients following the procedure. New pathological Q waves developed in leads V^1 and V^2 in three patients. Present data support the previous findings of the development of new anterior ST elevation, Q waves, QRS widening, RBBB, LAFHB, permanent heart block, transient QT interval prolongation. None of the ECG criteria showed statistically significant association with the measured echocardiographic parameters.

Key words: Hypertrophic cardiomyopathy, percutaneous, electrographic chage

INTRODUCTION

Percutaneous Transluminal Alcohol Septal Ablation (PTSA) of the hypertrophied septum for patients with Hypertrophic Obstructive Cardiomyopathy (HOCM) is an effective, safe, endovascular, non-surgical therapeutic intervention. By reducing the size of the asymmetrically hypertrophied septum, a significant decrease in the Left Ventricular Outflow Track (LVOT) dynamic gradient is noted. This improves heart failure symptoms. HOCM patients have an increased risk for malignant cardiac arrhythmias. There have been questions whether PTSA increases this risk by creating fibrous scar tissue in the septum. Additional complications of PTSA include extensive myocardial necrosis and high-grade heart block necessitating permanent pacemaker placement.

Multiple reports indicate clinical and hemodynamic benefits after PTSA in HOCM patients[1-3]. Previous studies attempted to correlate PTSA associated ECG changes with hemodynamic parameters in small number of patients. We report a larger series of patients with longer follow up. We also attempt to correlate ECG findings with echocardiographic measurements during the follow up period. Since HOMC patients are at increased risk for arrthythmogenic death, it may be important to attain additional risk stratification following PTSA in order to

Corresponding Author: Garbor F. Matos, Department of Medicine/Division of Cardiology, 2160 South First Avenue, Loyola University Health System, Maywood, Illinois 60153

risk stratify patients prior to intervention. We wish to report our experience with ECG changes following PTSA in HOCM patients.

MATERIALS AND METHODS

We retrospectively reviewed records of 25 consecutive patients who underwent PTSA for symptomatic HOCM in our tertiary center between 3/2001 and 5/2003. Offered PTSA to patients with symptoms of heart failure, angina or syncope refractory to medical therapy with beta blockers, verapamil or dual chamber pacing. Inclusion parameters included asymmetric subaortic septal hypertrophy, resting LVOT gradient exceeding 30 mmHg and signed written consent prior to PTSA.

Standard twelve lead ECGs were done pre and immediately following PTSA, as well as at follow up evaluations. The tracings were examined for rate, rhythm, PR interval, QRS width and axis, JT interval, R wave amplitude (in leads aVL and the lead with the tallest R wave), ST elevation, Q waves, atrio-ventricular, bundle branch blocks and QT/QTc interval.

Transthoracic echocardiography was done with Hewlett-Packard 5000 cardiovascular system using 2.5 MHz transducer. The images were analyzed on Kodak ImageVue version 3.02 imaging system for the dimensions of left ventricular cavity such as ventricular end diastolic diameter, atrial diameter and septal thickness, ventricular ejection fraction and ventricular mass. Left ventricular filling velocities and isovolumic relaxation time were measured from pulsed wave Doppler recordings of mitral inflow. LVOT gradient was determined by continuous wave Doppler tracings of the LVOT from apical five chamber or long axis views.

PTSA followed standard procedures, occluding the first and/or septal perforator arteries by a small balloon and injecting 1.5-5.0 mL^{-1} absolute alcohol over 3-5 minutes under continuous echocardiographic, ECG and hemodynamic monitoring. Records were reviewed for patient demographics, medical history and medications. Descriptive and statistical (paired Student-t tests) analyses were performed.

RESULTS AND DISCUSSION

There were 25 patients, 13 males and 12 females, with mean age of 58.5±11.7 (36-86) years with a mean follow-up of 164days±234.3 (2-783) days. Prior to intervention the mean class of NYHA heart failure symptoms was 2.8. Three (12%) patients had syncope and one (4%) patient had non-sustained ventricular tachycardia captured by Holter monitor. Medical therapy included beta-blocker 13 (52%), Verapamil 4 (16%) or the combination of beta blocker and Verapamil 2 (8%).

PTSA induced myocardial injury was documented by a creatine kinase peak at 1273.1±617.5 (618-2939) IU/mL^{-1}, creatine kinase-isoenzyme MB at 147.1±74.5 (76-275) ng/mL^{-1} and cardiac troponin I at 29.3±20.58(21-77) ng/mL^{-1}. There was a remarkable and sustained improvement in heart failure symptoms from the baseline NYHA functional class of 2.8±0.7 to 1.1±1.0 post-PTSA ($p<0.0001$). None of the patients experienced subsequent angina. One patient developed progressive heart failure and died of unknown cause 1-month post-PTSA. The patient devoloped no complications pre, peri or post-operatively. Peak creatine kinase patient was documented at 1528 IU/mL^{-1} and peak cardiac tropinin I of 34.5ng/mL^{-1} both occurring less then 3days post-PTSA.

The mean baseline LVOT gradient of 50.3±30.5 mmHg decreased significantly 65% to 17.8±14.3 mmHg at immediate follow-up, ($p<0.0001$). Patients with over 3-month follow-up maintained a low LVOT gradient of 18.3±12.6 ($p<0.0001$). Left ventricular mass of 244.6±94.0g was reduced 3% to 237.5±85.2g ($p<0.0045$) and 15% to 200.1±87.4g ($p<0.0086$) in this subgroup.

The hypertrophied segment of the interventricular septum of 3.8±0.8cm decreased 37% to 2.4±.5cm ($p<0.0001$), then 16% to 2.0±0.4 cm ($p<0.0640$) 3 months later. Left atrial diameter of 4.3±0.9 cm was reduced 15% to 3.7±0.8 cm ($p<0.0004$) and remained at 3.8±0.8 cm ($p<0.051$) 3 months later.

No significant change in resting heart rate or PR interval was recorded during the study period. Four (16%) patients required permanent dual chamber pacemaker implantation secondary to developing pacemaker dependent third degree heart block. Inclusion of these patients made no statistical changes to PR interval outcomes. Twelve (48%) patients developed permanent right bundle branch block, 6 (24%) patients left anterior fascicular hemiblock, 1 (4%) patients left posterior fascicular hemiblock and no patients left bundle branch block.

Baseline QRS interval duration of 113.4±32.1 msec increased 23% to 140.3±27.9 msec ($p<0.0001$) immediately post-PTSA. The R wave amplitudes did not demonstrate significant change during our study. Prolongation of the baseline QTc interval of 453.9±37.3 msec increased 8% to 491.3±52.6 msec ($p<0.0016$) (Fig. 1). No significant changes in JTc interval were noted from baseline to post-PTSA or at long term follow-up.

This suggests that the observed QTc interval prolongation was predominantly due to QRS widening.

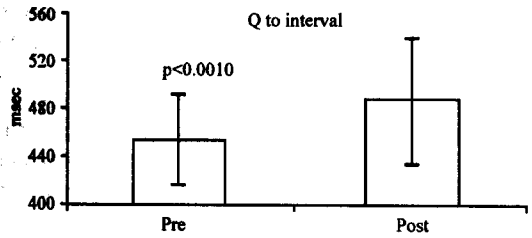

Fig. 1: Duration of QTc interval at pre-PTSA and immediately post-PTSA

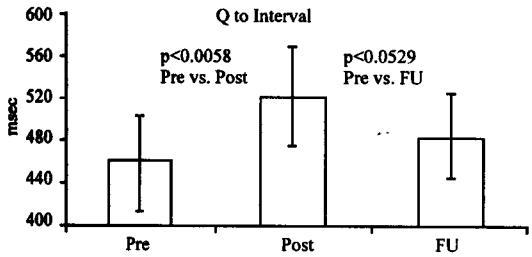

Fig. 2: QTc interval in the nine patient subgroup with long term follow up

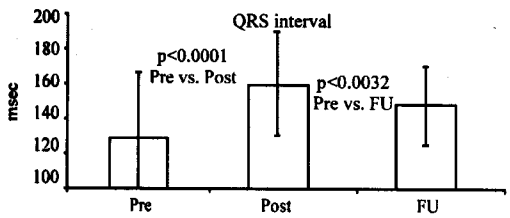

Fig. 3: QRS interval in nine patient subgroup with long term follow up

Three patients developed significant ST segment elevation of greater then 1mm in the anterior leads following PTSA, all of which resolved by follow-up examination. New pathological Q waves developed in leads V1 and V2 in 3 patients.

A subgroup of 9 (36%) patients had over a 3-month follow-up (360.0±260.3; range 108-783 days). Baseline QRS interval duration of 129.1±37.7 msec increased 24% to 160.7±30.8 msec ($p<0.0074$) at immediate post-PTSA, which decreased 7% to 149.1±2.7 msec ($p<0.0512$) at 3-month follow-up. Baseline QTc interval of 460.7±44.9 msec prolonged to 525.6±47.8 msec post-PTSA ($p<0.0058$). After 3 months QTc interval was 487.01±41.4 msec, not statistically different from baseline (Fig. 2). No significant JTc interval changes were noted from baseline to immediately post procedure or to long term follow up.

Present data are in agreement with existing reports demonstrating clinical, hemodynamic and echocardiographic long-term improvement of HOCM patients following PTSA[3-5]. A significant number of patients developed right bundle branch and/or hemi blocks post-PTSA. Our experience with the prognosis and natural course of the injured conduction system is predominantly based on the ischemic heart disease model.

The applicability of this model to the fibrotic, focal remodeling of abnormal myocardium in HOCM remains to be seen. The number of patients requiring permanent pacemaker placement is in agreement with current reports on post-PTSA complications[2]. Present study with a mean follow up of 164 days showed no adverse clinical effects in association with the development of bundle branch and or hemiblocks. Long-term follow up may be required for patients who developed PTSA associated conduction system abnormalities to monitor the development of progressive disease.

The increased probability for developing malignant arrhythmias in HOCM is a well-known risk factor and is responsible for sudden cardiac death in some of these patients. It is important to recognize the significant transient prolongation of the QT interval. To determine whether the transiently prolonged QTc increases the risk of malignant arrhythmias requires larger scale studies with longer follow up time. The unchanged JTc interval suggests that most of the QTc prolongation maybe attributed to prolongation of the QRS and not to abnormalities of repolarization. Since the clinical significance of this finding is currently unknown, the avoidance of any drugs with QT prolonging potential in the early post-PTSA period should be advocated.

Analysis of a 9 patient subgroup indicates improvement of the initial QTc interval prolongation three months after the procedure (Fig. 3).

CONCLUSIONS

PTSA of the hypertrophied septum in patients with HOCM results in impressive hemodynamic, echocardiographic and clinical improvements. These changes are also associated with potential complications, such as the development of bundle branch and heart block. The number of patients requiring permanent pacemaker in this study is in agreement with previously published data. The development of fascicular and bundle branch block did not predict the development of complete heart block at long term follow up in our patient population.

There is significant prolongation of the QRS and QT intervals following PTSA. Repolarization abnormalities generated by the alcohol induced acute infarct questions the possibility of increased malignant arrhythmia risk in this already high risk group. The stable, non prolongation of the JT interval suggests that the QT prolongation is predominantly due to lengthening of the QRS interval. Nevertheless, avoidance of QT prolonging medications

during the first three months following PTSA seems to be a safe practice.

The quantitative decrease in echocardiographic and hemodynamic results do not demonstrate a significant correlation with the observed ECG findings.

Longer follow up is required to assess the natural course of PTSA related conduction abnormalities and changes in the incidence of malignant arrhythmia. Such data could provide additional risk stratifications following PTSA. As a result, HOCM patients would be better informed of post procedural complications.

Since HOMC patients are at increased risk for arrthythmogenic death, it may be important to attain additional risk stratification following PTSA in order to risk stratify patients prior to intervention.

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